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Journal of Clinical Case Reports and Clinical Study

Open Access Case Report

Acute Severe Mitral Regurgitation due to Papillary Muscle Rupture after Blunt Chest Trauma: Case Report

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Article Info

Received: March 15, 2021
Accepted: March 22, 2021
Published: March 26, 2021

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Citation: Ibrahim Farah, Ali Abdelghaffar, Hanaa Elmahdy, Yasser Mubarak. "Acute Severe Mitral Regurgitation due to Papillary Muscle Rupture after Blunt Chest Trauma: Case Report". Clinical Case Reports and Clinical Study, 3(1); DOI: 10.61148/2766-8614/JCCRCS/039

Abstract

Cardiac injury is a common unexpected organ injuries leading to mortality in polytrauma patients. Cardiac injuries following blunt chest trauma vary from myocardial contusions to fatal rupture. **(Shaikh N., et al. 2013)**

Blunt cardiac injury is about 15—25% of blunt chest trauma. The most common form is cardiac contusion with variable manifestations accompanied by electrocardiogram *(ECG)* or cardiac enzyme abnormality. **(Yang S., et al. 2011)**

Traumatic rupture of intra-cardiac structures is an uncommon after blunt chest trauma; however there are few reports related to rupture of intra-cardiac valves. Valves involvement are uncommon, the most frequent being the aortic valve, followed by the mitral and tricuspid. Nowadays, there has been a rise in the incidence of these injuries due to increase of road traffic accidents *(RTA)*. **(Bernabeu E., et al. 2004)**

Rupture of papillary muscle or its chordae tendineae following blunt chest trauma is a very rare causing acute mitral insufficiency with subsequent congestive heart failure *(HF)* and pulmonary edema. **(Shaikh N., et al. 2013)**

Keywords: Blunt Chest Trauma; Acute Mitral Regurgitation; Papillary muscle rupture

Introduction:

Blunt chest trauma seldom results in cardiac injury, which varies in severity from asymptomatic myocardial contusion to fatal cardiac rupture **(Cresce GD., et al. 2009).** However, intra-cardiac injury incidence has been described aortic then mitral regurgitation seems to be extremely rare **(Bernabeu E., et al. 2004).** Blunt chest trauma is a rare cause of acute mitral regurgitation *(MR)*. Papillary muscle rupture caused by blunt chest trauma is a relatively rare cause of mitral incompetence **(Simmers T., et al. 2001)** (**Cresce GD., et al. 2009)**.

The mechanisms of injury could be the sudden increase of intra-ventricular pressure due to compression of the heart between sternum and vertebral column, or due to the sudden deceleration injury with the heart being pushed forward against the sternum **(Cresce GD., et al. 2009).** Sudden deceleration or compression of the heart with subsequent damage to the closed atrioventricular valve or subvalvular apparatus is responsible; patients are generally road traffic accident *(RTA)* victims **(Simmers T., et al. 2001).**

Traumatic mitral valve *(MV)* injury occurs during late diastole and early systole. The most common mitral injury is papillary muscles rupture, followed by the chordae tendineae, and a leaflet tear. Manifestations of traumatic mitral injury

injury is papillary muscles rupture, followed by the chordae tendineae, and a leaflet tear. Manifestations of traumatic mitral injury include a wide range of presentation varying from asymptomatic to acute cardiogenic shock. **(Bernabeu E., et al. 2004)**

Case report

We report a case of 27-year-old male patient with no previous medical diseases. He was complaining of shortness of breath *(SOB)* and stitching chest pain after road traffic accident *(RTA)* one week ago. His vital signs were Heart Rate 100 beats/minutes (tachycardic), Blood Pressure 100/70 mmHg, Respiratory Rate 35 breath/minute (tachypneic), Temperature 36.5°c, Glasgow Coma Score *(GCS)* 14/15, and O2 saturation 94% on 3Liters breath mask. On auscultation a significant pansystolic murmur was heard at apex propagated to axilla. Electrocardiography *(ECG)* revealed diffuse ST concave elevation. Laboratory data was Troponin I (0.011 ng/ml), lactate dehydrogenase *(LDH)* (1,54 mU/mL) and creatine phosphokinase *(CK)* (456 mU/mL). Arterial blood gas *(ABG)* analysis in a 3Liters breath mask were as follows: *pH* 7.35, carbon dioxide tension 42.5 mmHg, oxygen tension 84 mmHg, base excess of –2.0 mmol/L and oxygen saturation 94%. Chest X-ray showed diffuse opacity patches bilaterally without thoracic cage fracture or pneumothorax or pleural effusion. Chest computed tomography *(CT)* revealed bilateral infiltration. Covid-19 swab was done and result was negative. Transthoracic echography *(TTE)* revealed severe mitral regurgitation with fail anterior mitral leaflet and rupture of anterolateral papillary muscle ***(Figure 1,2)***. *EF* was 77%. Within twelve hours of admission to emergency department, he developed progressive worsening *SOB* and was transferred to intensive care unit *(ICU)*, where he received intravenous Furosemide and Nitroglycerin. Final diagnosis was traumatic rupture of anterolateral papillary muscle of *MV*, after blunt chest trauma, causing acute severe *MR* and pulmonary edema. He accepted surgery and transferred to cardiac surgery center. Then, emergency mitral valve replacement performed.

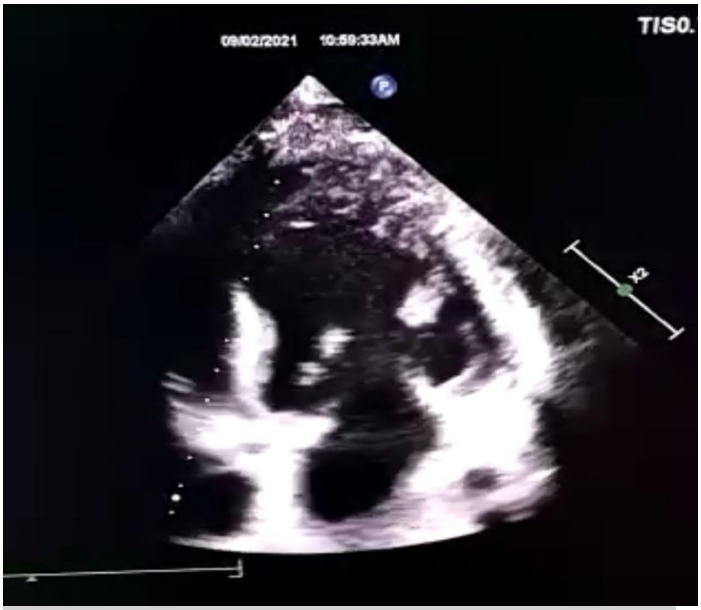

Figure 1: TTE revealed severe mitral insufficiency with rupture papillary muscle

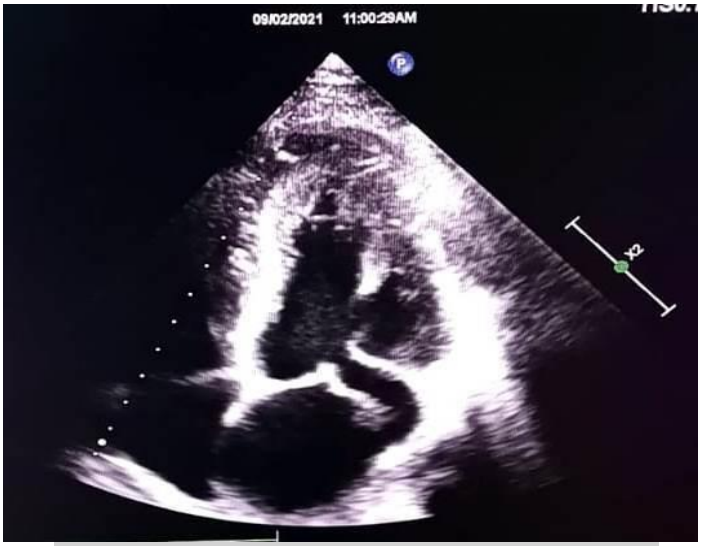

Figure 2: TTE revealed flail anterior mitral valve leaflet

The decision was made to operate, with the intention of repairing the valve, if possible. After induction, *TEE* was performed, confirming severe *MR*, together with an echogenic mass prolapsing into the left atrium *(LA)*. This mass was presumed to be the papillary muscle *(PM)*. After the institution of cardiopulmonary bypass *(CPB)* and cardioplegic arrest, *LA* was opened. The anterolateral *PM* had torn out of the left ventricular wall and twisted itself several times around the chordae. Because of the possibility of failure with simple re-attachment of the anterolateral PM to the friable ventricular wall, the *MV* was replaced with *St. Jude Medical* 29-mm mechanical valve ***(Figure 3)***. The atrium was closed, and the patient weaned off bypass easily. His postoperative course was uneventful, and an echocardiogram on the 5th postoperative day *(POD)* showed well function prosthetic *MV* with good *EF*. He was discharged to home the 10th *POD* after *INR* adjusted, and was seen in clinic 2 weeks later, at which time he was asymptomatic. 3-month follow-up period, clinical was well and asymptomatic, *TTE* revealed well function *MV* without gradient, and *INR* was 2.5 on warfarin 5mg.

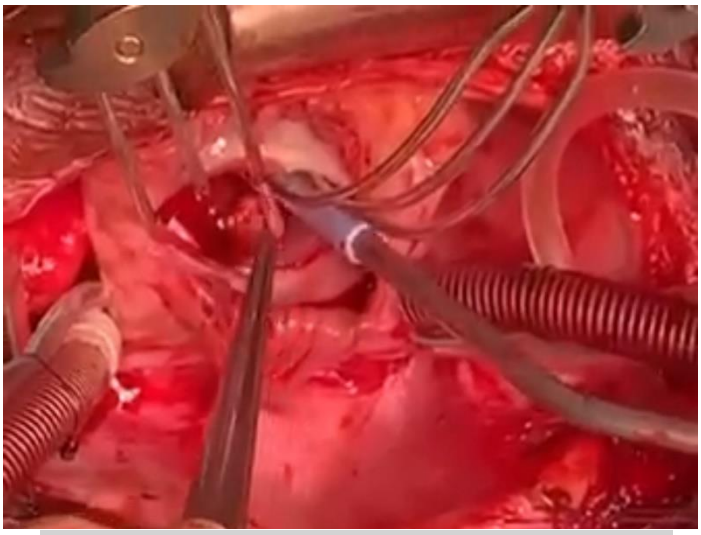

Figure 3: intra-operative photo of rupture papillary muscle

Discussion

Thoracic trauma is a common reason for presentation to the emergency services, especially following *RTA*. Myocardial contusion is frequent sequelae. However, acute valvular dysfunction following blunt chest trauma is a rare event **(Halstead J., et al. 2000)**. First mitral traumatic rupture was reported in 1936 and the first successful repair in 1964 **(McLaughlin JS., et al. 1964)**. Recently, the incidence of blunt chest trauma has been increasing because of traffic accidents, and a few cases unfortunately lead to death without appropriate diagnosis and treatment in spite of improved diagnostic procedures and surgical techniques due to lack of high index of suspicion. Generally, myocardial injuries are not uncommon, although *MV* injury is very rare. In cases of blunt chest trauma, it is clinically important to detect new systolic murmurs and any symptoms of heart failure *(HF)*, and to do urgent surgery. Ordinarily, *TTE* and *TEE* are used as diagnostic tests for this injury. Surgeons should determine whether to perform *MV* repair or replacement according to intraoperative findings. Mitral Valve Replacement *(MVR)* is a reliable and simple procedure. The choice for a particular approach must be based on the extent of damage, accurate analysis of the mitral apparatus and surgeon's technical expertise **(Bernabeu E., et al. 2004)**.

Traumatic acute severe *MR* is thought to occur as a result of loss of valve integrity from a sudden increase in intra-cardiac pressure while the heart is completing diastole and entering systole, with the ventricles dilated and the atrio-ventricular valves closed. Most frequently damaged is the papillary muscle, followed by the chordae tendineae. Symptoms of acute *MR* are due increased *LA* pressure and decreased left ventricular *EF*. Acutely, the patient usually presents with signs of pulmonary edema such as dyspnea, cough and orthopnea **(Petteys S., et al. 2011)**.

Blunt injury to the cardiac valves leads to progressive *HF* often requiring surgery. Most frequently, prosthetic replacement is the option of choice. *MR* following non-penetrating cardiac injury is usually the result of *PM* rupture, other causes being chordal rupture, or leaflet disruption. Papillary rupture can be partial occurring through one of the heads, or complete through the muscle body. In this latter case especially, severe left ventricular failure ensues and surgical management is usually essential. The presentation can be delayed, perhaps due to eventual rupture of a necrotic papillary muscle contused in the initial trauma **(Halstead J., et al. 2000)**.

Cardiac valve injury is an uncommon clinical entity, the recognition of which is difficult at the time of initial evaluation in cases of blunt chest trauma. Because the patient with traumatic valve injury often has hemodynamic instability, insufficient visualization of the friable endocardium, and necrotic *PM*, the valve repair is neither easy nor safe. Especially for *MV*, some authors have recommended that valve replacement should be done for all cases of acute valvular disruption. A high index of suspicion is important for the early diagnosis of traumatic valve injury. Even when cardiac valve injury is equivocal, one should not hesitate to perform *TEE* **(Choi JS, and Kim EJ. 2008).**

Traumatic *MR* if not detected early and treated properly can get complicated and progress to congestive *HF* and cardiogenic shock. This primary damage resulted in the development of coagulation necrosis, resulting in subsequent rupture. Patient's clinical condition will detect the timing and type of surgery **(Shaikh N., 2013)**.

Conclusion

In cases of blunt chest trauma, it is clinically important to detect newly developed systolic murmurs and any symptoms of congestive heart failure. Echography is essential in diagnosis. It is important to perform surgery in a timely fashion. Replacement of injured mitral valve is safe, rapid, and the suitable option in most cases.

Abbreviations

ABG: arterial blood gas, **CK:** creatinine kinase, **CPB**: cardiopulmonary bypass, **CT**: computed tomography, **ECG**: electrocardiography, **EF**: ejection fraction, **GCS**: Glasgow coma score, **HF**: heart failure, **INR**: international normalized ratio, **LA**: left atrium, **LDH**: lactate dehydrogenase, **MR**: mitral regurgitation, **MV**: mitral valve, **MVR**: mitral valve replacement, **SOB**: shortness of breath, **PM**: papillary muscle, **POD**: post-operative day, **RTA**: road traffic accident, **TEE**: trans-esophageal echo, **TTE**: trans- thoracic echo.

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